Basilar Artery Disease

Clinical Manifestations

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• **Patients with basilar artery disease show their first symptoms by characteristic ischemic attacks. If the attending physician correctly diagnoses the condition and initiates anticoagulant therapy before a catastrophic episode occurs, there is reason to believe that the disease may be arrested temporarily.**

IN RECENT YEARS basilar artery disease has received a great deal of attention from neurologists as well as other physicians who are interested in cerebral vascular conditions. The classical description of this syndrome was given by Kubik and Adams[1] in 1946. It has been further documented by reports from the Mayo Clinic[3,4] as well as from neurologists in several other centers in the United States.

The present report is of 35 cases of basilar artery syndrome in which the patients were observed by the authors or in which the clinical summaries and reported neuropathological observations were felt to be of sufficient interest to warrant including them.

The series included 22 males and 13 females ranging in age from 42 to 90 with an average of 62 years. Twenty-four of the patients had blood pressure higher than 145/90 mm. of mercury and 11 also showed evidence of having generalized arteriosclerosis, which may or may not be associated with the two previously mentioned disease entities.

Seventeen patients gave a clear history of ischemic attacks; and a history suggestive of ischemic attacks was obtained in other patients but it could not be clearly substantiated before a thrombotic episode and death.

The criteria for anticoagulant therapy are (1) a cooperative patient; (2) no medical disease that would contraindicate the use of anticoagulants; (3) a physician who is familiar with the management of patients receiving anticoagulants; and (4) blood pressure below 190/110 mm. of mercury. The patient and the family should be informed about the possible side effects as well as the rationale for the use of the therapy.

Eight patients were treated with anticoagulants and at last report were getting along very well, without recurrence of attacks. In two other cases complete thrombosis of the basilar artery occurred despite anticoagulant treatment and the patients died. A high proportion of the patients in the series had ischemic attacks, which often were not recognized by their family physicians, for as long as four years before the occurrence of a thrombotic episode. For this reason a report of some of the typical ischemic attacks as described by patients in the group studied would appear worthwhile.

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REPORTS OF CASES

CASE 1. A 55-year-old hypertensive white woman described many episodes of severe vertigo occurring almost daily for four years before the occurrence of an acute thrombotic episode. Three months before she entered the hospital she had a severe episode of vertigo which was followed by deafness in the right ear. Severe vertigo also occurred at the time of the thrombotic episode, which left her with multiple palsies referrable to the cranial nerves, severe dysarthria and bilateral long-tract signs. At last report the patient was in a rest home receiving terminal care.

CASE 2. A 42-year-old white man, observed in neurological consultation, had a history of periodic alternating hemiplegias, blurring of vision and severe dysarthria. Quadriplegia developed while he was in the hospital, and administration of anticoagulants was immediately started. At last report he had been doing well for two years and had returned to work. Bilateral carotid arteriograms in this patient were entirely within the limits of normal.

CASE 3. A 44-year-old white man gave a history of having two types of ischemic attacks: (1) Mental confusion and almost complete loss of vision in both eyes, lasting from 2 to 5 minutes, (2) severe vertigo with quadriparesis. After repeated attacks in the period of a year, anticoagulants were administered. Two weeks after this therapy was begun the patient suddenly died and at autopsy a pontine hemorrhage was observed. Pronounced calcification in the basilar and vertebral arteries was seen in x-ray films of the skull.

CASE 4. A 50-year-old hypertensive white man gave a history of having had severe attacks of vertigo, circumoral numbness, dysarthria and weakness of the left arm and leg daily for three months. One typical attack was witnessed by the author who noted that during the attack the patient had vestibular nystagmus, pronounced dysarthria, depressed corneal reflexes, left-sided pyramidal tract signs and slight mental confusion. These signs disappeared within five minutes, except for the depressed abdominal reflexes and the left extensor plantar response. When last observed the patient had been receiving anticoagulant therapy for four months and was free of ischemic attacks.

CASE 5. A 57-year-old white man complained of periodic episodes of dizziness, diplopia and unsteady gait for five months. At times he had as many as ten of these attacks a day. Three months before entering the hospital the attacks began to be associated with circumoral numbness. The day before entering, he had an episode of severe vertigo, nausea and vomiting and numbness of both the upper limbs. The final day, complete occlusion of the basilar artery occurred after one of the typical spells, and pathological examination confirmed the clinical impression.

CASE 6. The patient, a 65-year-old woman, had had repeated attacks of vertigo, dysarthria, diplopia, numbness of the right side of the face and paraparesis lasting from 1 to 5 minutes for five days before she was observed in consultation. When last observed, she had been receiving anticoagulant therapy for three months and had had no more ischemic attacks. An x-ray film of the chest showed pronounced calcification of one aortic arch.

Headache was one of the most common symptoms in patients with ischemic attacks as well as in those who had occlusive episodes without a definite history of neurologic disease. The headache was usually in the suboccipital region and, while not severe, often was painful enough to cause the patient to use an analgesic drug. Very often headache preceded the thrombotic episode. Some investigators believe that headache in such cases is secondary to dilatation of the external carotid artery.[2] Very frequently the terminal thrombotic episode began with suboccipital headache.

Paralysis of various types, involving two, three or four limbs was also commonly noted in patients with basilar artery occlusion. Visual disturbances, ranging from almost complete loss of vision in both eyes to slight blurring of vision in one or both eyes, were extremely frequent. Diplopia in one or more than one field of gaze was observed in 13 of the 35 patients. Tinnitus was not a common symptom. Sensory disturbances of various types were elicited in the history taking, the most common being circumoral numbness and numbness of one side of the face and the opposite side of the body. Twenty-three of the 35 patients gave a history of vertigo, generally speaking not associated with tinnitus. Only one patient reported loss of hearing.

Conjugate gaze palsies or impairment referrable to the third, fourth or sixth cranial nerve on one or both sides were frequently noted. Of special interest was the case of a 55-year-old woman with a history of hypertension, rheumatic and arteriosclerotic heart disease who had a sudden episode of syncope lasting five minutes. When she awoke, bilateral third nerve palsy was noted. She died later of a massive occlusion of the right middle cerebral artery, and at autopsy cystic changes were observed in both oculomotor nuclei. Visual field defects, including altitudinal field defects and cortical blindness, were noted in seven patients. In a number of cases adequate examination of visual fields could not be carried out. Bilateral facial and pharyngeal weakness or paralysis was noted in 25 patients. The corneal reflexes were characteristically absent or depressed in the great majority of the patients who died or were under terminal care at the time of this report.

Sensory examination of severely ill patients was of little help in establishing a diagnosis. In the case of patients who were having ischemic attacks, the history of circumoral numbness or paresthesias involving one or both sides of the body was undeniably one of the factors in establishing a diagnosis.

The motor system was involved in 34 of the 35 patients, and history of bilateral tract involvement was given by all of the 25 patients who died or were under terminal care at the time of this report. Dysmetria and dysdiadochokinesia were also quite frequently noted; but in the case of severely ill patients with pronounced involvement of the pyramidal tract, observation for these signs was not feasible.

Either unilateral or bilateral extensor response to plantar stimulus was noted in all patients in the group who died or were under terminal care. Abnormality of abdominal reflexes was also noted in all such patients. A tendency to plantar response was noted also in patients who were having minimal ischemic attacks, even in the period when they had no symptoms.

Three of the patients became akinetic and mute but were able to understand spoken directions.

Calcification in the carotid, basilar or vertebral arteries, or in branches of the thoracic aorta, were observed in six patients.

Spinal fluid examination was carried out in 26 cases. In 13 of them the protein content was above 50 mg. per 100 cc. The highest value obtained was 164 mg. In one patient the cerebrospinal fluid pressure was 240 mm. of water, the only case in which pressure exceeded 200 mm.

Two of the patients died while receiving anticoagulant therapy; in one of them (previously mentioned) death was believed attributable to the drug. The other patient, after being symptom-free for about a year, suddenly became quadriplegic, anarthric and comatose at his home. He died and at autopsy thrombosis of the basilar artery was noted. One hypertensive and diabetic patient who refused anticoagulant therapy was living and well at the time of this report, two years after onset of symptoms, except for periodic attacks of vertigo.

In electroencephalograms, obtained in six patients, no specific pattern was noted, only generalized slowing.

All but two of the 21 patients who died were examined neuropathologically at autopsy. Of these patients, eight had the thrombotic lesion at the level of the junction of the vertebral and basilar arteries. Five of them had thrombosis in the distal end of the pons with resultant infarction in the distribution of the posterior cerebral arteries and superior cerebellar arteries. In the remainder of the cases there was rather diffuse thrombosis with areas of infarction extending throughout the distribution of the basilar artery. In the cases discussed in this paper, it will be seen that these ischemic attacks are fairly classical. The physician who makes such a diagnosis should institute therapy, if there are no contraindications.

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REFERENCES

1. Kubik, C. S., and Adams, R. D.: Occlusion of the basilar artery—a clinical and pathological study, Brain, 69:73-121, 1946.

2. Meyer, J. S., Sheehan, S., and Bauer, R.: An arteriographic study of cerebrovascular disease in man; 1. Stenosis and occlusion of the vertebral-basilar arterial system, A.M.A. Arch. Neurology, 2:27-45, Jan. 1960.

3. Millikan, C. H., and Siekert, R. G.: Studies in cerebrovascular disease: I. The syndrome of intermittent insufficiency of the basilar arterial system, Proc. Staff Meet. Mayo Clin., 30:61-68, 1955.

4. Millikan, C. H., Siekert, R. G., and Shick, R. N.: Studies in cerebrovascular disease: III. The use of anticoagulant drugs in the treatment of insufficiency or thrombosis of the basilar artery, Proc. Staff Meet. Mayo Clin., 30:116, 1955.

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